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Occupational Health in Ontario

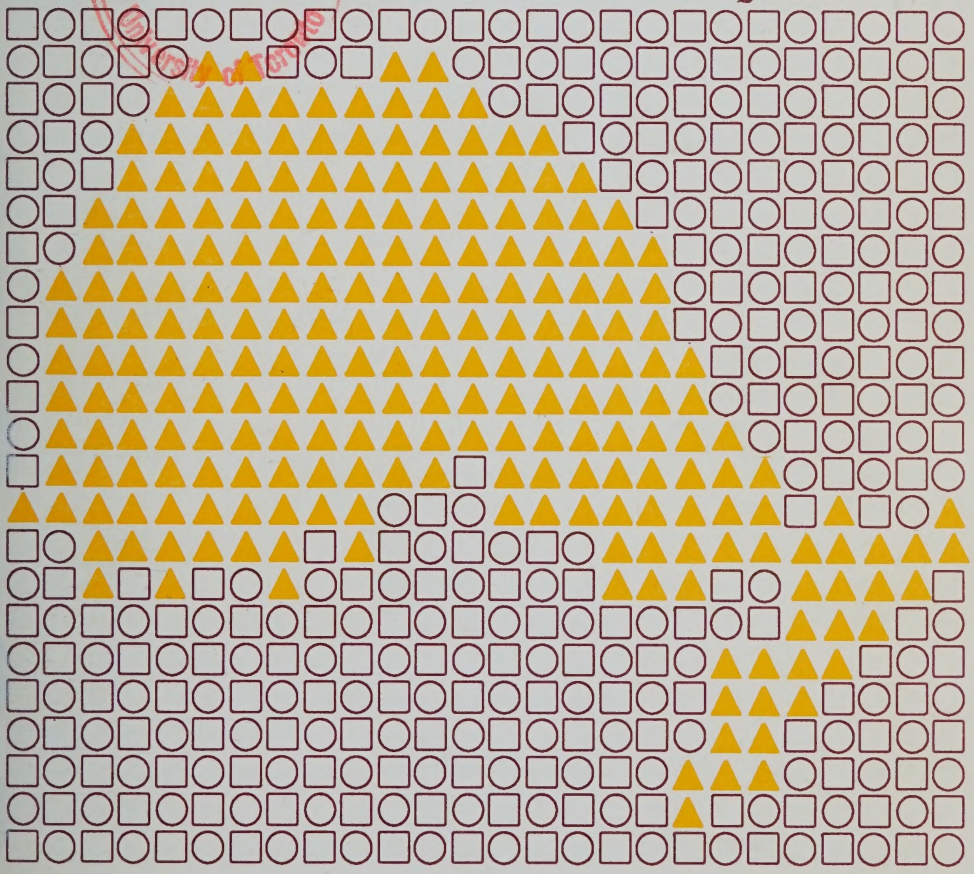
Volume 12, Number 2, Spring/Summer 1991

Mixer and Applicator Exposures to Lawn Spray Chemicals

Occupational Asthma: A Review

Occupational Health Hazards in Hospitals

Administration of Indoor Air Quality Questionnaires



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Occupational Health in Ontario

Volume 12, Number 2, Spring/Summer 1991

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3. U.S. Department of Health, Education and Welfare. Smoking and Health. A report of the U.S. Surgeon General, 1979.

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Correspondence

To the Editor

I read in the article by Ronald E.M. Lees entitled 'Ontario's Occupational Health Resource Centres After 12 Years' (Volume 12, Number 1, Winter 1991) that 'the Canadian Centre for Occupational Health and Safety has recently been forced to eliminate its free on-line inquiry service' and that 'the user fee now in place has reportedly reduced the number of inquiries, resulting in a 66 per cent drop in on-line computer connections to the CCOHS' (p. 16).

This statement is misleading and inaccurate; what Mr. Lees refers to in his article is the fact that in 1990 CCOHS started charging for connections to CCINFOline, its computerized information service, which provides a direct link to the CCOHS host computer. CCOHS's computerized information service is also available on compact disc read-only memory (CD-ROM) through a subscription to CCINFOdisc. Verbal advice remains available, free of charge, to anyone in Canada. As noted further down in the same publication (p. 33), CCOHS continues to 'offer a free, confidential Inquiries Service ... available on a toll-free line across Canada.'

The Inquiries Service handles daily 100 requests for information on occupational health and safety and remains very much a part of CCOHS business strategy for the coming years.

I would appreciate your making your readers aware of the inaccuracy in that article.

J. Arthur St. Aubin
President and Chief Executive Officer
Canadian Centre for Occupational Health and Safety

Dr. Lees' response to Mr. St. Aubin is printed below.

To the Editor

My statement, referred to in Mr. St. Aubin's letter, was referenced, the source being an article by W. List in *Canadian Occupational Safety.* In his letter Mr. St. Aubin admits that CCOHS is now charging for its on-line computerized information service, but does not confirm that there has been a reduction in use, as claimed by W. List. If there has been no fall in the on-line (to my mind that means direct access to computer) information service of CCOHS, then the source article cited by me was in error. If that is the case, I regret that I unwittingly relayed this misinformation. My intent was to deplore that diminishing government support for free occupational health and safety information is impeding access to a vital public service.

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Mixer and Applicator Exposures to Lawn Spray Chemicals

Summary

Hygiene assessments of mixer and applicator exposures to lawn spray chemicals (i.e. pesticides and fertilizers) were made at nine of 18 local lawn-spraying companies. These companies represent a cross-section of the lawn care industry, ranging from owner-operated businesses with one or two seasonal workers to a large, international company with thousands of employees. The inhalation potential is considered minimal, and respiratory protection is generally not necessary. With the exception of

Key Words: lawn spray, pesticides, herbicides, insecticides, fungicides, fertilizers, 2,4-D

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The most commonly used pesticides for lawn spraying are the chlorophenoxy herbicides, 2,4-D and mecoprop; the phthalic acid derivative, chlorthal-dimethyl; and the organophosphate insecticides, chlorpyrifos and diazinon, with lesser quantities of dicamba and minor use of various others. The human toxicity of these pesticides in concentrated form is low to moderate by inhalation, skin absorption or ingestion,[1-3] and the hazards are further reduced by dilution prior to spraying (typically less than 0.5 per cent total pesticide content, and almost always less than 1 per cent). The active pesticide ingredients are non-volatile and become airborne as dusts or mists. However, the organic solvent or oil carriers in some pesticide concentrates (notably chlorpyrifos formulations) are detectable by odour and can gen- erate significant airborne vapour concentrations in a small, poorly ventilated area, especially during hot weather.[1-4] The question of 2,4-D carcinogenicity continues to be studied by the National Cancer In- stitute in the United States,[3,5,6] but the U.S. Environ- mental Protection Agency and the Ontario and New Brunswick environment ministries have concluded that existing uses of 2,4-D pose no significant health risk.[5]

During the summer of 1989, local concerns about potential occupational exposure of lawn spray applicators to pesticides and fertilizers were brought to the attention of municipal and regional government agencies. The concerns expressed were for the short- and long-term health effects to employees because of the perceived inadequacy of personal protective clothing and equipment. The present study details the results of an occupational hygiene survey, carried out by the Ontario Ministry of Labour, of representative lawn-spraying companies in southwestern Ontario.

Procedure

Visits were made by an occupational hygienist to nine of 18 active lawn-spraying companies in the region to assess worker exposure to lawn spray chemicals. The companies, selected to represent a cross-section of the lawn care industry in this region, ranged from owner-operated businesses with one or two seasonal workers to a large, international company with thousands of employees.

inadequate footwear worn by applicators at one company, chronic exposure of both mixers and applicators by skin contact was adequately controlled by personal protective equipment; mixers, however, were generally not fully protected against accidental skin or eye contact with concentrated pesticides. Applicators may face increased exposures from occasional drifting of spray, or skin contact with spilled material or contaminated equipment. Recommendations are made for upgrading hygiene and emergency response facilities, and for drift control procedures and personal protective equipment.

Results and Discussion

Lawn Spray Chemicals

Lawn spray chemicals used by the nine companies are indicated in Table 1. Fertilizers, when used, are non-volatile, relatively low toxicity materials, such as urea or potassium nitrate, or blends of urea, ammonium polyphosphates, potassium chloride or similar substances. Three companies visited apply only granular fertilizers, and six include a fertilizer at about 2–5 per cent concentration in various herbicide and insecticide formulations.

Mixing

Mixing of lawn formulations is done in delivery truck tanks of 425- to 3,000-litre capacity or, occasionally, in 15- to 20-litre back-pack tanks. Practices vary with respect to the order of ingredient additions, but generally, water and fertilizer are added first, followed by pesticide concentrates and recirculation of the tank contents. Most often, solid fertilizer is dumped into the top opening of the tank from 10–25 kg bags, although two companies pumped in liquid fertilizer pre-mix from bulk storage. Pesticides are always added manually: liquids are measured and transferred in open volumetric containers of up to 10-litre capacity, and solids are poured from pre-weighed bags or are manually weighed and added. The liquid measuring containers are filled from 200-litre drums by gravity (cradle and bung-valve) or manual drum pump, or from 2- or 10-litre containers by pouring.

Without exception, these operations are done

Table 1	Lawn Spray Chemicals Used								
	Company Number								
	1	2	3	4	5	6	7	8	9
Fertilizers									
18–2–4 formulation (N,P,K)	*								
Urea		*					*		
Potassium Nitrate				*					
35–5–10 formulation						*			
14–3–2 formulation								*	
Herbicides									
Mecoprop Amine	*	*	*	*	*	*	*	*	*
2,4–D Amine		*	*	*	*	*	*	*	*
Dicamba	*			+	*	*		*	
Chlorthal–dimethyl (Dacthal^{R})	*		*			*	*	*	
Bensulide (Betasan^{R})				*	*				
MCPA	*								
Glyphosate (Round–up^{R})	+	+		+		+			
Fenoxaprop–ethyl (Acclaim^{R})			+	+	+	+	+		
Insectidides									
Chlorpyrifos (Dursban^{R}), (Fosban^{R})	*	*	*	*	*	*	*	*	
Diazinon	*						+		+
Malathion				+					
Fungicides									
Benomyl (Benlate^{R})	+			+				+	
Chloroneb (Tersan^{R})				+				+	
Iprodione	+								
No. of Employees:	40	10	20	4	4	3	2	4	6

*Major Use (>50L or kg per year)
+Minor Use (<50L or kg per year)

outdoors or in a large, open area. Pesticide concentrate additions generally involve less than 4.5 litres, which the worker adds by climbing onto the truck bed or platform. One of the companies using bulk liquid fertilizer pre-mix has incorporated a pesticide eductor (aspirator) into the transfer system. This practice permits pesticide concentrates to be added from the main floor level, thus reducing the likelihood of a spill or splash.

In some cases, mixing is done by the lawn spray worker, but generally, the handling of pesticide concentrates is restricted to specified management personnel. The concentrates are typically handled for a few minutes, although sometimes up to half an hour, once or twice per day. Personal protective equipment varied widely, but all mixing personnel wore neoprene or nitrile gloves to prevent skin contact with the hands. At one company, rubber boots and a rubber apron, a helmet, a face shield and an organic vapour respirator were also worn, with chemical-protective goggles optional. At the other end of the scale, another company's workers used only the gloves.

Lawn Spray Applicators

Lawn spray applicators varied from students with one or two weeks' field training to full-time, licensed specialists, with the majority being experienced seasonal workers. Spraying is almost entirely done between March and October, principally in the spring and early summer, and again in the late summer and the autumn. Hours are long, typically 11 or 12 hours a day, six days a week during peak periods. The applicators spray 20–40 average lawns (3,000–4,000 square feet) per day, with an actual spraying time of 10–15 minutes each.

Practices

Dilute lawn spray formulations were delivered by various pump, hose and nozzle systems, all of which directed a spray towards the ground in front of the applicator, from hip-height or lower. Most (seven of nine) of these systems were designed to produce relatively large droplets to minimize chemical 'drift',

and one system employed a wand arrangement with four nozzles that produced a finer spray, but from a height of only one–two feet above the ground. One small company, however, to improve coverage, purposely used a fan spray nozzle with a relatively small droplet size. Although control of drift was recognized as an obvious concern by all nine companies, only one had an established maximum permissible wind–speed policy; that company has equipped its applicator personnel with pocket wind–speed indicators, and, because of excessive drifting of spray with its equipment, spraying is discontinued when the wind speed exceeds 25 km per hour. At the end of each spray application, the hose was manually or mechanically rewound, being directed back and forth onto the reel by hand or, in one case, with a special tool.

With two exceptions, lawn spray applicators were required to wear rubber boots, long trousers, a shirt, and neoprene or nitrile gloves as the minimum level of personal protective equipment. One exception, at the company where manual handling of contaminated hose is avoided, was the absence of gloves. The other involved use of leather work boots in place of rubber boots. Some companies provided organic vapour/mist respirators for voluntary use, and these were usually carried in the truck cabs. Management reportedly audits the use of personal protective equipment by field workers, at least on a spot–check basis, at eight of the companies visited, although records of these checks are not generally maintained.

Two companies have documented ongoing training and testing programs, and one of these firms requires all its field personnel to become licensed pesticide applicators. Licensing is done by the Ministry of the Environment under the *Pesticides Act* and Regulation 751, which classify pesticides according to their toxicity and define legal requirements for licensing and for the safe storage, handling, use and disposal of pesticides. Each lawn spray company is required to have a Class 6 Operator's Licence for land exterminations and is required as well to employ at least one licensed land exterminator for every

three or fewer employees who assist in performing exterminations. In addition, pesticides are controlled products under WHMIS (Workplace Hazardous Materials Information System) legislation, and all workers who may be exposed to these substances must be trained in their hazards and in appropriate control measures. Pesticides are currently exempt from WHMIS labelling and material safety data sheet requirements, although this exemption is under review.

To facilitate good hygiene and emergency response capabilities, all lawn spray trucks at one company have been provided with soap, water, towels, first-aid kits, material safety data sheets, written emergency procedures, and such spill containment equipment as shovels, sorbent pillows and storm sewer drain covers. Practices among the other eight companies vary, but few carry more than data sheets and, perhaps, a first-aid kit.

Medical Surveillance

Medical surveillance was generally voluntary and left to the discretion of individual workers, although most companies advised pre-season examinations and cholinesterase baseline determinations. One company also advised mid-season testing, a policy with which at least some of its employees comply, and one provides initial and regular cholinesterase determinations every three weeks throughout the spraying season and maintains records of the results. The voluntary nature of worker participation in medical surveillance is in keeping with human rights legislation in Ontario and with section 17(3) of the *Occupational Health and Safety Act* (as amended by Bill 208), which states that a worker is not required to participate in a prescribed medical surveillance program unless the worker consents to do so.

Exposure Risk

Since the active ingredients in lawn spray formulations are non-volatile, they do not become airborne except as dusts or mists. Some pesticide concentrates contain organic solvent or oil carriers

that are detectable by odour and may generate significant vapour concentrations in a small, poorly ventilated area, but mixing and loading take place outdoors or in large, open areas. Spraying, of course, generates dilute aerosol mists, but the risk of inhalation is considered minimal, owing to the position of the nozzle relative to the worker's breathing zone. Consequently, respiratory protection is considered unnecessary for most mixing and spraying.

Respirators that were provided for voluntary use were, with some exceptions, appropriately selected. However, they were usually stored unprotected with other items in locations such as truck cabs and were not cleaned and disinfected after use; consequently, worker exposure to contaminants may actually have been increased by respirator use, rather than decreased.

The pesticides can be absorbed through the skin and, ordinarily, this is the most significant route of exposure for both mixers and applicators.[1, 7-12] Workers at all companies visited wore neoprene or nitrile gloves to prevent chronic skin contact, but the potential for accidental skin or eye contact in case of a spill or splash was not completely prevented. In eight cases, the likelihood of a spill or splash was increased because workers had to climb onto the trucks to access tank openings. Concentrated fertilizers, which also have some potential for skin or eye irritation, are considered less hazardous than the pesticides and were adequately controlled by existing measures.

The principal exposure risk to lawn spray applicators is via contact with the skin of the feet and lower body during spraying, and with the hands when rewinding contaminated hoses or through accidentally contaminated gloves or spilled material.[7, 8, 9, 12] In all but one case, where leather work boots rather than rubber were worn, adequate personal protective equipment was worn to prevent chronic skin contact. Most of the companies, however, had the potential for occasional increased exposures from chemical drift or accidental contact with spilled material or contaminated equipment. The following recom-

mendations were developed to further reduce worker exposure to pesticides during mixing and spraying:

1. To protect against accidental skin or eye contact when handling concentrated pesticides, mixers should wear chemical–protective goggles, a face shield, an impervious apron and impervious gloves and boots. Consideration should also be given to reducing the likelihood of such accidents by improving the transfer process (e.g. using a pump or eductor system to eliminate the need to climb onto trucks; providing easier access to the tanks).
2. As a minimum, lawn spray applicators should wear rubber boots, long trousers, a shirt and, where handling of contaminated hoses is unavoidable, neoprene or nitrile rubber gloves.
3. Chemical drift during spraying should be minimized by using as coarse a spray as possible from the lowest spray height, establishing a maximum permissible wind–speed policy and determining wind speed prior to spraying.
4. To facilitate good hygiene practices and emergency response capabilities, lawn spray trucks should carry soap, water, towels, first–aid kits, and spill containment equipment (e.g. shovels, sorbents and storm sewer drain covers).
5. An appropriate method should be developed and used for directing contaminated hoses onto the reels without manual contact during rewinding.
6. Where respirators are provided for voluntary use, a respirator program should be implemented. This program should include fit–testing, proper storage and worker training in proper respirator cleaning, maintenance and use.

Conclusions

Overall health and safety programs of the companies visited varied widely, but in most cases advice was given to upgrade hygiene and emergency response capabilities and to minimize drifting of spray mists. Although only one of the nine companies conducts regular biological monitoring to ensure the adequacy of its control measures, the controls in place were generally adequate and the health of workers is considered unlikely to be adversely affected by mixing and/or applying lawn spray chemicals.

Résumé

L'évaluation sanitaire de l'exposition des mélangeurs et des épandeurs de produits chimiques d'entretien des gazons (c.-à-d. antiparasitaires et engrais nébulisés) a été menée dans 9 des 18 entreprises locales d'épandage des gazons. Ces entreprises représentent une vaste section transversale de l'industrie de l'entretien des gazons, variant de l'entreprise exploitée par le propriétaire et comptant un ou deux travailleurs saisonniers, jusqu'à la grande entreprise internationale comptant des milliers d'employés. Le risque d'inhalation est jugé minime et, en règle générale, la protection des voies respiratoires n'est pas nécessaire. À l'exception de chaussures inadéquates que portaient les épandeurs dans une des entreprises, l'exposition chronique des mélangeurs et des épandeurs par contact cutané était contrée adéquatement par l'équipement individuel de protection; par contre, les mélangeurs n'étaient généralement pas totalement à l'abri de contacts accidentels d'antiparasitaires concentrés avec la peau ou les yeux. Chez les épandeurs, il peut y avoir un risque accru d'exposition par dérive occasionnelle du produit nébulisé et contact avec la peau en raison de produits déversés ou de matériel contaminé. Les auteurs formulent des recommandations pour l'amélioration des installations d'hygiène et des mesures d'intervention d'urgence, des modalités de contrôle de dérive et du matériel de protection individuelle.

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Occupational Asthma: A Review

Summary

Occupational asthma is a disorder characterized by reversible airways obstruction, caused by factors in the work environment. Several pathogenetic mechanisms have been described and an extensive number of etiologic agents have been identified. The evaluation of suspected cases of occupational asthma requires both a careful review of the clinical presentation and the judicious use of such clinical investigations as skin tests and serology, pulmonary function measurements and bronchial provocation tests. The diagnosis of occupational asthma is made when the relationship between the asthma and the working

Key Words: occupational asthma, pulmonary function, airways, sensitization, allergy

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Asthma is a common disease affecting large numbers of people. It is estimated that 8 to 10 million Americans have asthma.[1] Between 2 and 15 per cent of all cases of asthma are of occupational origin.[1,2] The pathogenesis, etiologic causes and diagnostic techniques have been well characterized. Diverse views exist, however, on the definition and classification of occupational asthma.

Definition

Asthma is generally defined as recurrent episodes of wheezing or dyspnea characterized by variable airways obstruction, reversible spontaneously or by treatment.[1,3] Occupational asthma, then, is asthma due to factors in the work environment. Some authors limit the definition of occupational asthma to that which occurs because of sensitization to a substance encountered at work.[3,4,5] Others have a broader definition and consider working exposure to any airborne dusts, gases, vapours or fumes.[6,7] Most will not consider workers with pre-existing asthma aggravated by the workplace as having occupational asthma, on the basis that the work environment has not caused the asthma.[3,5]

The definition of occupational asthma and its distinction from non-occupational asthma is important for diagnostic, therapeutic and medico-legal reasons. The definition of occupational asthma may be broadened for purposes of workers' compensation (where there may be a benefit-of-the-doubt policy) and where mandatory reporting of sentinel cases to government health departments is required.[8] A definition for the purposes of an epidemiologic investigation may have to be precise, with detailed diagnostic criteria specified.

Pathogenesis

environment is established. Early diagnosis of occupational asthma, combined with removal from exposure, leads to a more favourable prognosis.

A number of pathogenetic mechanisms of asthma have been described by various authors. Morgan has described the pathophysiological responses of the respiratory tract to particles, mists and gases.[9] Bronchoconstriction occurs where the site of deposition is the trachea and bronchi. The mechanisms involved are immunologic, pharmacologic and irritant or reflex. Other, less well-defined mechanisms may also exist.

Salvaggio et al., in their work for NIOSH,[1] include all three mechanisms in their description of occupational asthma, as does the Society of Occupational Medicine in Great Britain.[10] Warren and Hargreave[4] and the Canadian Thoracic Society[5] limit their definition of occupational asthma to include the immunologic mechanism only, and they specifically exclude non-immunologic mechanisms from the definition.

The immunologic mechanism is the most commonly described. This form of asthma is then called either allergic asthma[2] or occupational immunologic lung disease (sub-type: asthma).[11]

Examples of agents (known as allergens or sensitizers) causing allergic asthma are given in Table 1. According to the Canadian Thoracic Society, more than 300 substances may cause allergic asthma.[5] Most allergenic agents causing asthma are high molecular weight compounds such as laboratory animal dander and urine, plant dusts and enzymes. These compounds induce allergic bronchoconstriction by the production of specific IgE and sometimes IgG antibodies.[12] Low molecular weight compounds such as colophony, metal salts, isocyanates, anhydrides and plicatic acid in western red cedar dust act as haptens, combining with serum proteins to produce an allergen. The ultimate result of IgE production from either high or low molecular weight compounds is an immediate-type hypersensitivity response (Type I response). This develops in a variable number of individuals, following a latent period in which sensitization takes place. Subsequent exposure to low concentrations of the

antigen then results in bronchoconstriction.

Irritant–induced variable airways obstruction is well described in the literature. Some authors, however, differentiate it from occupational asthma and consider the latter to be only of immunologic origin, by definition. Brooks et al.[13] described a persistent asthma syndrome after exposure to high levels of irritants, which they termed Reactive Airways Dysfunction Syndrome (RADS). They ascribed a non–immunologic mechanism, involving bronchial epithelial damage, and concluded that this asthma syndrome is different from typical (immunologic) occupational asthma.

Saric has described a syndrome of asthma–like symptoms and signs induced by respiratory irritants, based on observations of potroom workers.[14] Saric includes this syndrome in the rubric of occupational asthma. The postulated pathogenesis is abnormalities in the autonomic regulation of airway smooth muscles with alteration of the balance between vagal and sympathetic stimuli. Boulet studied increased bronchial responsiveness following acute exposure to respiratory irritants (sulfuric acid, hydrochloric acid and bleaching agents) and differentiated this from increased bronchial responsiveness following exposure to sensitizing agents (perchloroethylene and toluene diisocyanate), terming the former RADS (or high–level irritant induced asthma) and the latter occupational asthma.[15]

Tarlo and Broder, in a study of 154 workers with occupational asthma, concluded that irritant–induced occupational asthma has features similar to occupational asthma caused by sensitizers. They consider irritant–induced occupational asthma to be a form of occupational asthma caused by single or multiple exposure to irritants.[16]

The pharmacologic mechanism of asthma is mediated through activation of the complement system, resulting in the release of histamine and other substances. Both the classical and alternative pathways may be involved. The alternative pathway may be non–immunologically activated by organic dusts such as grain dust,[17] resulting in an asthmatic

Table 1	Examples of Agents Causing Occupational Asthma
Agent	**Industry**
High molecular weight compounds	
Mammalian residues	Laboratory workers, veterinarians
Bird residues	Farmers, bird breeders, poultry workers
Plants (grain, flour, vegetable fibres)	Grain handlers, bakers, brewers
Enzymes (B. subtilis, trypsin)	Detergent industry, pharmaceutical industry
Arthropods (insects, mites, crustacea)	Laboratory workers, grain workers, crab processing
Molluscs	Oyster farming
Low molecular weight compounds	
Isocyanates	Painters, polyurethane foam manufacturing, foundries
Epoxy resins (tetrachlorophthalic anhydride, trimellitic anhydride)	Plastics, adhesives
Metals (platinum, nickel, chromium)	Refining, plating, tanning
Soldering flux (colophony)	Electronics industry
Wood dust (western red cedar, oak, mahogany)	Sawmills, carpentry
Drugs (penicillin, cephalosporins, psyllium)	Pharmaceutical industry
Formaldehyde	Insulation, adhesives, disinfectants, tanning, plastics

Modified from Chan-Yeung and Lam.[6]

response. In addition, the complement system may also be immunologically activated, resulting in occupational immunologic lung disease, mentioned above. Isocyanates have been reported to exert a pharmacologic effect by acting as beta receptor blocking agents.[1,18]

Some agents may act through a combination of the above mechanisms, or through other, unknown mechanisms. For example, asthma due to isocyanates has been postulated as being caused by the allergic mechanism,[12] the irritant mechanism[16] and the pharmacologic mechanism.[6]

Clinical Presentation

Patients with occupational asthma usually present with complaints of cough, chest tightness, wheezing and dyspnea.[19,20] The respiratory symptoms are often accompanied by rhinitis and/or conjunctivitis. Cough, sputum and chest pain are present in about two-thirds of patients with occupational asthma, which may result in an initial diagnosis of bronchitis if these are the main presenting features.

The symptoms of asthma occasionally occur at work and frequently occur maximally within several hours after leaving work. Three types of asthmatic reactions have been described by Pepys and Hutchcroft[12] and other authors.[19] These include: the immediate reaction, where symptoms develop within minutes of exposure at work; the late reaction, where delayed symptoms occur several hours after exposure, during the evenings of workdays (and not at all on days away from work or on vacations); and the dual reaction, where both immediate and late symptoms occur. Work-related changes in asthma medication requirements may have similar patterns. Workers with severe occupational asthma may only improve with an extended stay away from work, or may not improve at all. In a study of 125 patients with asthma due to western red cedar, Chan-Yeung et al. report that 9.6 per cent of patients presented with an immediate reaction alone, 43.2 per cent with a late reaction alone and 47.2 per cent with a dual reaction.[19] Late asthmatic responses are considered as characteristic of allergen-provoked rather than

irritant-provoked asthma.

Asthmatic symptoms may develop after a worker starts a new job or after new materials are introduced on a job. There may be a substantial period between the initial exposure and onset of symptoms, during which time sensitization has occurred. There may also be an interval between onset of exposure and onset of symptoms in irritant-induced asthma. Tarlo and Broder reported on three patients with irritant-induced asthma who had exposures at work for over six months prior to onset of symptoms.[16] Brooks et al., however, in their diagnostic criteria for RADS, required onset of symptoms within 24 hours of the exposure.[13]

A variant of the clinical presentation described above may occur with asthma secondary to vegetable fibre dusts such as cotton, flax, hemp or jute. The syndrome is known as byssinosis. Classically, patients present with Monday morning chest tightness, with symptoms occurring several hours after returning to work from a weekend or vacation. The symptoms improve overnight and occur with lessening severity later in the workweek. Histamine release and subsequent depletion over the workweek has been postulated as the mechanism of action.[6]

The physical examination of a patient with occupational asthma may be normal, or there may be wheezes or rales, depending on the pattern of the asthmatic response and the severity and stage of the illness.

Clinical Tests

Skin Tests and Serology

Skin testing may be helpful both in assessing the atopic status of the worker and for testing for the presence of specific IgE antibody against an occupational agent. Skin testing is usually done with the epicutaneous method (the prick test). If the prick testing is negative, then intradermal testing is used.

The suspected allergen, when injected into the skin, reacts with IgE antibody on the surface of mast cells or basophils. These cells then release agents

such as histamine and 5-hydroxytryptamine, producing the classic wheal and flare reaction. Common aeroallergens and food allergens are utilized in prick testing, for determination of atopic status.[19]

Skin tests for high molecular weight occupational agents such as those present in laboratory animal dander, plant dusts and enzymes are often positive in sensitized individuals, since specific IgE is produced against the complete molecule. Low molecular weight chemicals that act as haptens must be conjugated to human serum albumin to provide a usable skin test preparation. Skin test reactivity has been found useful for only a few low molecular weight agents, such as platinum[20] and nickel salts, and anhydrides.[21] Skin testing carries a small risk of a serious reaction or of sensitization in a non-allergic individual. Medications may interfere with skin reactivity. In vitro serologic testing avoids these two problems.

Various in vitro serologic tests are available to detect IgE and non-IgE antibodies (IgG, IgA, IgM). The radioallergosorbent test (RAST) is a commonly used test for detecting the presence of specific IgE antibodies directed against a particular antigen. In this test, antigen bound to a solid phase reacts with antibody in the patient's blood, which in turn is bound by radiolabelled antiglobulin. The amount of bound radiolabel, determined by scintillation counter, is proportional to the amount of antigen-specific IgE. RAST testing has the disadvantage of being more expensive and less sensitive than skin testing. Chan-Yeung reported that specific IgE antibodies to crude red cedar extract or plicatic acid-human serum albumin conjugate as determined by RAST, were found in fewer than 40 per cent of patients with western red cedar asthma.[22] Another technique, enzyme-linked immunosorbent assay (ELISA), has been used in the determination of specific IgE and IgG to human serum albumin conjugates of phthalic anhydride and tetrachlorophthalic anhydride.[21]

When performing skin testing and serology, one must remember that positive tests by themselves only indicate exposure and sensitization to an agent, and do not necessarily indicate the presence of asthma. A positive test is only suggestive evidence of a causal clinical relationship to a suspect agent. In addition, it has also been shown that positive skin tests may not develop until after respiratory symptoms have developed.[21] Specific skin tests, therefore, have little prognostic value for the development of occupational asthma. Whether this is also true of serology is unknown. A diagnosis of occupational asthma, therefore, cannot be made by skin tests and serology alone.

The solutions for IgE–specific skin testing and the antibody–specific serologic tests are generally available only in specialized asthma and allergy centres. Antibody tests have been developed for only a very limited number of agents.

Additionally, as the mechanism of asthma is often not allergic, skin and serology tests have a limited role in the clinical investigation of asthma.

Pulmonary Function Measurements

Pulmonary function testing may be useful in investigating workers with continuing exposures to suspect agents. It is important to note, however, that in many asthmatics a single measurement may be normal on a day away from work. Normal tests, therefore, cannot be used to exclude a diagnosis of occupational asthma. The most simple application of pulmonary function tests is to monitor for a decrease in FEV_1 (forced expiratory volume in one second) before and after a workshift. However, although this method may detect workers with an early asthmatic response, it will miss late reactors. The best day to measure pre– and post–shift FEV_1 is the first workday of the week, as the pre–shift FEV_1 will gradually decrease with successive workshifts over the workweek, resulting in a lessening of the pre– and post–shift change at the end of the week. The post–shift FEV_1 can also be repeated 10 minutes after the administering of a bronchodilator, and one

should look for reversibility of bronchoconstriction. Additional comparisons can be made between the FEV_1 (and symptoms and medication use) while the patient is at work and when he or she has been off work for several days.

A significant reduction in FEV_1 over a single shift is generally considered to be 10 per cent or greater.[23] Some authors, however, consider a 15 per cent reduction in FEV_1 as the criterion for documenting variable airways obstruction.[24]

Changes in airway variability over an extended period can be detected by measuring peak expiratory flow rates (PEFR). Peak flows are useful to monitor while a patient continues at work, stops work and then resumes work. Burge recommends a minimum record of two weeks at work and two weekends away from work.[20] Another approach is to record peak flows for two weeks at work, two weeks off work and then another two weeks at work. This is known as a 'stop–resume' work test,[6] during which the patient's daily symptoms, medication requirements and peak flows are recorded. Burge recommends measuring peak flows every two hours while awake, using a portable peak flow meter. This scheduling may be somewhat demanding on the patient; therefore, others will conduct monitoring of peak flows every three hours[25] or three times a day (upon waking, after work and before bed).[6]

There are no universally agreed–upon criteria for interpreting changes in peak flows. Smith et al. diagnose variable airways obstruction if the difference between the minimum and maximum PEFR on at least one day exceeded 20 per cent of the day's maximum PEFR.[25] A reduction of 20 per cent in peak flows is a commonly utilized criterion,[23] although the required number of days for the reduction to occur varies among authors.[16] Problems with peak flow recordings include worker compliance, recording errors (both deliberate and accidental) and false negative results from concurrent use of inhaled corticosteroids or disodium cromoglycate.

Non–Specific Bronchial Provocation Tests

Two types of bronchial provocation tests are available for investigating suspected occupational asthma: non–specific testing and specific testing. In both tests, FEV_1 is measured prior to and after exposure to the test agent. The changes in FEV_1 are analysed.

Non–specific bronchial provocation tests are performed to measure bronchial hyperreactivity. Patients with asthma demonstrate bronchial hyperreactivity to various non–specific stimuli that produce bronchoconstriction. Methacholine (or histamine) inhalation challenge testing is used to elicit this hyperreactivity. The test is simple, safe and reproducible. The methodology of this test has been described by Lam et al.[26] Patients are instructed not to take any aerosol or oral bronchodilators for at least 12 hours prior to the test. Smokers are asked to refrain from smoking for at least two hours before the test. Test solutions for inhalation are aerosolized by a nebulizer with oxygen. The patients breathe the aerosol via a loosely fitting face mask and breathe with normal tidal volume.

The first solution inhaled is buffered saline solution, which serves as a control, followed by increasing concentrations of methacholine (starting with 0.03 mg/ml, to maximum of 24 mg/ml). Solutions are given for two minutes. After each inhalation, the FEV_1 is measured at 30 seconds and four minutes. The inhalations are carried out at five–minute intervals until the FEV_1 has fallen by at least 20 per cent below the lowest post–saline value, or until 24 mg/ml has been given. If the patient reacts to methacholine, the FEV_1 is repeated at five, 10 and 15 minutes after the last inhalation. The percentage fall in FEV_1 is calculated using the lowest post–saline FEV_1 as the baseline value. The provocative concentration of methacholine required to produce a 20 per cent fall in FEV_1 from the baseline value is known as the PC_{20}. This value is interpolated from data points of the inhalation test, plotted as per–centage fall in FEV_1 versus methacholine con–centration. A methacholine PC_{20} of 8 mg/ml or less

indicates bronchial hyperreactivity.[16]

A four–fold improvement in methacholine PC_{20}, after removal from workplace exposures, is considered significant.[16] Where the PC_{20} is greater than 16 mg/ml, it is unlikely that the patient is asthmatic. Therefore, the test is useful in differentiating asthma from chronic bronchitis, as the PC_{20} is generally in the normal range with chronic bronchitis.

It should be noted, however, that an asthmatic patient who has been away from workplace exposure and has become asymptomatic may have a normal PC_{20}. Lam et al. have shown that non–specific bronchial hyperreactivity in asthmatics will decrease towards normal after removal from exposure, and will increase following re–exposure to the offending agent.[26]

Specific Inhalation Provocation Tests

Methacholine challenge testing by itself will not identify or confirm the etiologic agent involved in a case of asthma. Specific provocation tests are indicated in the following instances: where it is necessary to determine the precise etiologic agent of asthma (e.g. when studying previously unrecognized inducers of occupational asthma and for guidance in job placement and agent avoidance for workers exposed to several agents known to cause asthma); when required for medico–legal or compensation purposes; and if peak flow measurements and methacholine challenge tests are confusing or, because workplace exposures have been already discontinued, not applicable. Inhalation provocation testing with workplace substances is potentially dangerous and should be performed by experienced personnel in a hospital setting where resuscitation facilities are available and where frequent observations can be made over sufficient time to monitor for late reactions and, where necessary, treat them.

The methods used in specific provocation testing have been described by Pepys and Hutchcroft.[12] Testing is performed either by aerosolization and

inhalation of the specific agent, or by exposing the patient to a simulated working environment. The most sophisticated test will have a precise control of exposure levels and will be carried out in a whole-body exposure chamber. General principles involved in testing include only exposing the patient to subirritant levels of chemical agents and not to exceed normal workplace exposure levels. The object of the test is to establish the lowest exposure concentration capable of eliciting a definite, reproducible reaction. The test result shows whether the particular agent is capable of provoking the subject's symptoms. Because it utilizes subirritant exposures, this test is not used in the diagnosis of irritant-induced asthma.

Initial test concentrations of the suspect agent to which the patient is exposed, along with the exposure time, is guided by the clinical history, the known toxicology of the test agent and the patient's degree of bronchial reactivity on non-specific provocation testing. Patients with low levels of bronchial reactivity to methacholine can be challenged with higher concentrations of the test agent, for a longer time interval.

For the aerosol inhalation test, solution containing the test agent is nebulized with oxygen or air. The patient inhales the aerosol through an oro-nasal mask. Aerosolization of powdered agents can be achieved by pouring containers of the powder back and forth, although the exposure levels are not well controlled in this method.

Simulation of the working environment may involve having the patient perform painting or other appropriate activities with the workplace mixture of the test agent, rather than with the pure form.

Generally, baseline pulmonary function tests are performed prior to challenge testing. Challenging is performed on one day with a control, and on another day with the test agent itself. The patient is blinded to the control and the agent tests. Control testing is necessary to exclude non-specific reactions. Before provocation testing is performed, it may be necessary to remove workers from exposure for several weeks

to eliminate non-specific reactions.[20] Bronchodilator medications are withheld prior to testing, since these may inhibit both the immediate and the late responses.

The inhalation tests start with a brief exposure period of 30 to 60 seconds, followed by a 10-minute interval during which the FEV_1 is measured. Where there is no clinical reaction or change in FEV_1, the test is repeated. Either the exposure concentration or the exposure duration can be increased in subsequent testing. A positive challenge test is considered to be the development of asthma symptoms and an accompanying decline in FEV_1 greater than 20 per cent compared with baseline and control values.[27]

A positive provocation test following exposure to subirritant levels of the test agent leads to a straightforward diagnosis of occupational asthma due to the agent. Negative provocation testing may occur in a patient with true occupational asthma because of technical factors in the test, such as wrong particle size, incorrect temperature of the agent, or missing antigens (where the asthma may be due to a different workplace agent).[20] A negative test, therefore, does not exclude a diagnosis of occupational asthma.

Evaluation of Work Environment

Patients often present to physicians unaware of what chemicals are in their workplace or of the extent to which they are exposed to them. A thorough evaluation of the workplace by an industrial hygienist may be necessary to determine the presence of sensitizers and irritants in the workplace; to determine levels of exposure; to evaluate accidental exposures; and to advise companies on primary prevention through controlling exposures to workplace chemicals. Data on workplace exposures are often crucial to the determination of the etiologic agent for a case of asthma. This information is also necessary to assist in job placement of affected individuals.

Data on exposure levels to sensitizing agents should be interpreted with the understanding that there is little information on level of exposure that results in

sensitization. Exposure levels that trigger an asthmatic response in a sensitized individual are usually much lower than levels required to induce sensitization. As government–regulated exposure standards to chemical agents are generally set to protect healthy non–sensitized individuals, workers with allergic asthma may react to levels well below these standards.

Predisposing Factors

Several factors, such as atopy, smoking and pre–existing bronchial hyperreactivity, have been suggested as playing a role in the development of occupational asthma.[1,4,6,16,19,28] However, the significance of these factors and their mechanism of action have not been well established. Workers, therefore, should not be denied employment based on the presence of predisposing risk factors. They should be counselled about the potential slight increase in risk.

Prognosis

Several studies have shown that many patients with occupational asthma do not recover completely after cessation of exposure to sensitizing agents such as western red cedar,[19,22] tetrachlorophthalic anhydride[29] and isocyanates.[30] Chan–Yeung et al. found that patients with persistent asthma had a significantly longer duration of symptoms before diagnosis, had lower lung function test results, and had a more severe degree of non–specific bronchial hyperreactivity at the time of diagnosis than did those who recovered.[19]

Symptoms of irritant–induced asthma may also persist for months to years following cessation of exposure, or they may persist with continuing exposure.[16] Prognostic factors, though, have not been as well characterized for irritant–induced asthma as they have for allergic asthma.

It is apparent, however, that early diagnosis of any type of occupational asthma, together with removal from exposure, may lead to a more favourable prognosis. In some workers, though, the asthma may persist after removal from exposure to the etiologic agent, but the severity of the asthma will decrease.

Workers with occupational asthma who continue in exposure will likely experience deterioration of lung function.[19]

Management

Once the diagnosis of occupational asthma has been established, the worker should be removed from exposure.[4,5,6] This process will usually require consultation with and the co–operation of the affected worker, the employer, the union and Worker Compensation authorities. Ideally, the workplace should be modified to eliminate exposure to the offending agent. Sensitized individuals reacting to trivial exposures may, however, require a change of jobs. Workers with irritant–induced asthma may be able to continue at the workplace if exposures are minimized. Pharmacologic management of occupational asthma, with a combination of xanthine derivatives, beta–adrenergic agonists and corti– costeroids, does not differ from that of non– occupational asthma.

Physicians are often called upon to make an evaluation of impairment and disability for work placement or compensation purposes. Impairment is considered as the physiologic deficit reflected by pulmonary function tests, bronchial reactivity tests and medication requirements. Disability is a reduction of the capability to perform work or the activities of daily living. Determination of the degree of disability requires an evaluation of both the impairment present and the expected requirements of a job.[4,5] Because lung function may deteriorate slowly over time and job requirements may change, physicians should, on a periodic basis, reassess the impairment and disability of patients with occupational asthma.

Prevention

Prevention is classified as primary, secondary or tertiary. Primary prevention is the implementation of workplace controls to avoid having workers develop asthma. Various methods of control are possible. Elimination of hazardous materials — removing sensitizing agents from the workplace and substituting them with less hazardous or non–hazardous

materials, for example — is ideal, but not always practical. Other control strategies include isolating and enclosing a hazardous process; changing a process (to reduce airborne generation of contaminants); ventilating a process (to reduce levels of airborne contaminants); and using protective equipment. When evaluating workplace controls, one should remember that there are very few studies relating the level of exposure of a worker to sensitizing agents and the subsequent development of occupational asthma.[6]

Secondary prevention of occupational asthma would require the identification of susceptible workers and their removal from exposure prior to the development of asthma. This may be accomplished through periodic screening of workers for changes in pulmonary function. Serial measurements of FEV_1 may identify long–term effects of exposure. Changes in FEV_1 over a long period would be suggestive of an adverse effect. Annual decrements of 5 per cent or 0.2 litres should lead to further investigation.[18] The positive predictive value of tests of sensitization, such as specific skin tests and RAST tests, is sufficiently low for the development of occupational asthma to limit their value as screening tools for secondary prevention.

Tertiary prevention is the management and treatment of workers with occupational asthma, as discussed above.

Notification of cases of occupational asthma to appropriate health authorities, as occurs with the NIOSH (National Institute for Occupational Safety and Health) SENSOR (Sentinel Event Notification System for Occupational Risks) project, may identify workplaces requiring the establishment of primary preventive measures.[8] The diagnosis of a sentinel case of occupational asthma may also be the event that reveals the need to search for other cases, for remediable workplace factors and for recognition of new causes of occupational asthma.[23]

Discussion

Occupational asthma is a disorder characterized by reversible airways obstruction, caused by factors in the work environment. Pathogenetic mechanisms of occupational asthma include immunologic, pharmacologic and irritant–mediated bronchoconstriction. A combination of mechanisms or other, as yet uncharacterized, mechanisms may also be involved.

Clinically, patients complain of cough, chest tightness, wheezing and dyspnea. The symptoms may occur in relationship to exposure, with early, late and dual responses, accompanied by improvement on the weekends and during vacations. The diagnosis of occupational asthma is made by confirming the diagnosis of asthma and establishing a relationship between the asthma and the working environment. Tests available include pulmonary function tests, skin and blood tests for specific antibody, non–specific bronchial hyperreactivity tests, and specific inhalation provocation tests. The extent of testing required depends on the strength of the occupational history (exposure and symptoms), on whether the worker is continuing in exposure or not, and on the necessity for defining the etiologic agent of the asthma for job placement or compensation purposes. In certain cases, demonstrating a pre–shift to post–shift decrease in FEV_1 may be sufficient. In other instances, specific inhalation challenge testing will be necessary. For example, a worker with isocyanate–induced asthma who has been removed from the workplace may show FEV_1 changes only when challenged with isocyanate. Demonstration of sensitization by specific antibody tests is, by itself, insufficient for a diagnosis of occupational asthma.

Both primary prevention of occupational asthma (through workplace controls) and tertiary prevention (pharmacologic treatment) are well characterized. Secondary prevention, through serial measurements of FEV_1, may lead to early recognition of pre–clinical asthma, which, combined with appropriate intervention, may result in a more favourable prognosis. Further research is required on the role of

other prognostic factors.

Notification of sentinel cases of occupational asthma to health authorities may lead to identification of other cases, to the recognition of new causes of occupational asthma, and to the implementation of exposure control measures in a workplace.

Résumé

L'asthme professionnel est un trouble caractérisé par une obstruction réversible des voies aériennes, obstruction causée par des facteurs présents dans le milieu de travail. Les auteurs décrivent plusieurs mécanismes pathogènes et identifient un grand nombre d'agents étiologiques. L'évaluation des cas où l'on soupçonne l'asthme professionnel exige à la fois un examen prudent de l'aspect clinique et l'utilisation avisée d'enquêtes cliniques comme les tests cutanés et la sérologie, les mesures de la fonction pulmonaire et les tests de provocation bronchique. Le diagnostic de l'asthme professionnel s'impose lorsque le rapport entre l'asthme et le milieu de travail est établi. Si l'asthme professionnel est diagnostiqué tôt et que l'exposition est éliminée, le pronostic est plus favorable.

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Occupational Health Hazards in Hospitals: An Overview

Summary

Hospitals are a complex mix of clinical, laboratory and hotel services. The resultant array of health risks for staff requires the application of modern principles and practice of occupational medicine/nursing, hygiene and rehabilitation.

Key Words: hospital, biohazard, occupational medicine, hygiene, psychosocial hazard

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Introduction

In spite of alternate models of health-care delivery, including walk-in clinics, comprehensive health organizations and chronic-care facilities, active treatment hospitals continue to be the most visible elements in the overall system. They range from the basic community facility providing essential medical, surgical and obstetrical services to the thousand-bed tertiary-care medical school teaching centre. Whatever their size, hospitals represent a complex integration of clinical programs and industrial processes and are a major source of employment. The development of new modalities of investigation and treatment, supported by technology ever more sophisticated and complex, has characterized hospital progress.

The traditional healers—doctors and nurses, as well as the plethora of '-ists' (from psychologists to perfusionists)—have been accorded an exaggerated sense of responsibility for self-health and self-safety. Hospitals generally have been perceived to function at a higher level of employee safety than has industry. The notion of a concentration of clinical consultants committed to patient care is not unreasonable, but the assumption that clinical skills will just naturally protect both the workplace (hospital) and the non-patients (employees) has become suspect, particularly as viewed by organized labour.

Traditionally, the responsibility for radiation safety was left to the radiologists, concern for anaesthetic gases remained with the anaesthetists, and infection control belonged to the microbiologists. The historical concept of medical expert benevolence has been the rule rather than the exception. In fact, there continues to be a lack of physicians with formal occupational health qualifications committed to the

quality of life for the thousands of health-care workers in the hospital workplace. In Metropolitan Toronto, for example, only two hospitals support a full-time occupational medical director: Wellesley Hospital/Princess Margaret Hospital, and The Toronto Hospital/Toronto General and Western Divisions.

In addition to the lay assumption that hospitals are inherently healthful places in which to work, there are other factors that have inhibited the development of health and safety programs. The 'treatment-of-disease' orientation has tended to pre-empt competing interests in preventive medicine and health maintenance, while preoccupation with the patient's care and environment took precedence over the people providing such care. Professional and technological staff were assumed to be self-sufficient in the management of their own occupational health, while support staff often had little guidance at all. The availability of spontaneous informal medical opinion and/or treatment often aborted the reporting process of work-related health concerns and hindered the need for a more organized response. Finally, hospital funding, whether from government, direct patient accounts or endowments, has always been committed to direct patient care along with the other twin priorities of teaching and research. Only during the last decade have funds been more willingly allocated to employee occupational health and the monitoring of environmental biohazards.

In Ontario, hospital unions formerly pursued monetary and benefit considerations. However, the occupational health and safety legislation has established a link between hospitals and the Ministry of Labour, and has caused unions to focus on occupational health issues in hospitals.

One of the difficulties in addressing occupational health in hospitals is the diversity of the work done —the multiple services, both clinical and support, collectively reflecting a complex organization that never rests and whose demands span a huge range of human capability. The physical plant is similar to a

hotel of comparable size, but complicated by unique areas such as operating rooms, laboratories and imaging departments. The patient population varies from critically ill to terminally ill, and from pre-operatively well to post-operatively convalescent. The occupational health hazards will differ for those supplying the support services—engineering/maintenance, housekeeping, dietary—and for those providing the special services—nurses, physio-therapists and technologists. In the support areas, the environmental and physical hazards leading to injury are common to other industries having machine shops, mechanical equipment and food preparation functions. And then, of course, there is a whole range of biohazards that are the direct result of patient care, investigation and treatment.

Physical Hazards

Hospital physical plants may be compromised, if not by design then certainly by an insatiable need for space. An enclosed materials-receiving area, while protecting employees from wind, rain and cold, may simultaneously expose them to carbon monoxide from exhaust emissions as delivery trucks manoeuvre at the loading dock. Inefficient materials management and inadequate storage result in corridor clutter, often contravening fire codes.

Electrical hazards develop with the indiscriminate use of extension cords and also when staff bring in electric appliances for personal comfort and convenience—toasters, kettles and heaters, for example.

Noise levels from printing presses and dishwashing and laundry equipment may approach potentially harmful levels. Laundry functions have tended to become a supplied service from outside the hospital, but this change was based more on fiscal considerations than in response to any occupational health issue. Housekeeping staff are exposed intermittently to high noise levels from certain floor-cleaning equipment. Thoughtless placement of air conditioners, while providing comfort for some individuals, causes noise discomfort for others.

Hot environments within the hospital may include mechanical rooms and food service areas, especially during summer months, and can result in symptoms of heat exhaustion. Steam is a potential hazard for staff handling steam–sterilizing equipment, especially if such equipment is defective or improperly used.

Ionizing radiation has long been recognized as a hazard with the potential for tissue necrosis, malignancy, and teratogenic effects during pregnancy. Awareness of radiation safety in the modern hospital is essential in view of the profusion of imaging techniques. There is now virtually no part of the human anatomy that cannot be visualized by means of invasion, contrast media or radiation. Not only are radiologists and technologists at risk but so are cardiologists and orthopaedic surgeons using image intensifier equipment. Personal dosimeter badges should be worn by nurses and clinicians in these areas as well as by those health–care professionals in the traditional X–ray roles. The development of nuclear medicine and diagnostic scans has added the handling of radioactive materials to the growing list of hazards.

Various types of non–ionizing radiation sources can be found in hospitals, among them visible, ultraviolet and infrared light as well as ultrasound and microwave. Fluorescent and incandescent lighting are visible forms of radiation that may result in glare, especially in association with video display terminals.

Lasers (light amplification by stimulated emission of radiation) are used in microsurgery, in measuring immunoglobulins and other blood elements, and in imaging departments to assist in aligning patients for specific procedures. The skin and eyes are most susceptible to damage by lasers, which focus great amounts of light energy on a small surface area.

Ultraviolet radiation is emitted by some air filters, nursery incubators and germicidal lamps. Skin and eyes can be affected by overexposure. Infrared effects may occur from warming or heating equipment used in food preparation and during thermography. Damage occurs primarily as a result of a temperature rise in the absorbing tissue, causing

skin burns and adverse ocular effects. Microwave applications include sterilization of ampoules, diathermy and enzyme inactivation of tissues in certain research and cancer treatments. The heating of deep body tissues by microwave radiation may produce alterations in cells, resulting in possible neurological, behavioural and immunological changes. Ultrasound is used therapeutically, surgically and diagnostically. Injury to peripheral nervous and vascular structures can occur at points of contact with powerful ultrasound sources.

Chemical Hazards

The list of chemicals and chemical products in hospitals is seemingly interminable, with additions much more common than deletions. Housekeeping staff are exposed to the allergenic and irritant effects of soaps, detergents, polishes and disinfectants. Similar substances affect operating room nurses and attendants. Engineering/maintenance workers in buildings with older electrical installations may have to handle liquids containing PCBs, with resultant anxiety reactions. In the laboratory, chemicals range from potent acids to alkalis and include such specific items as o–toludine and benzidine for the testing of occult blood in stool specimens. Derived from the aromatic hydrocarbons toluene and benzene, respectively, these substances have been held responsible for methemoglobinaemia and hemorrhagic cystitis. In addition, they have been incriminated in malignancies of the urinary bladder even after latency periods of up to 40 years.

In the autopsy room and the surgical pathology laboratory, pathologists and technologists are exposed to formalin, with its irritant effects on eyes and on mucous membranes of the nose and respiratory tract. Repeated exposure may cause sensitization, resulting in asthmatic reactions at levels of exposure that are too low to cause symptoms in most people. Both primary irritation and sensitization dermatitis can occur. Formaldehyde is considered a suspect human carcinogen.

Food preparation and distribution imply the use of pesticides for fumigation and insect control. These

products have the potential for mishandling, spills and splashes, with resultant exposure.

The photography department has its own list of chemicals, and staff are unsurprisingly anxious when they are called upon to photograph clinical conditions of an infectious nature.

Anaesthetic gases have long been present in the ambient air of operating rooms, delivery rooms and recovery rooms. Nitrous oxide, halothane and penthrane have been detected in the expired air of anaesthetists and O.R. nurses, in amounts directly related to the hours of exposure. Proper technique by anaesthetists is essential to maintain a closed circuit between machine and patient; only then can scavenging systems work efficiently. Female health-care personnel exposed to waste anaesthetic gases may have a greater risk of spontaneous abortion. The wives of male operating room personnel may also have a higher incidence of miscarriage. Not only have hepatic and renal dysfunction been related to anaesthetic gases, but so have CNS effects, including headache, fatigue, irritability and sleep disturbances. Any relationship to malignant disease requires further long-term study.

While mercury thermometers have been replaced by higher technology in many hospitals, accidental spill and exposure may still occur as a result of broken mercury-containing blood pressure equipment. Inhalation is the usual route of entry, although skin absorption may occur as well.

Ethylene oxide exposure occurs when handling the sterilizing equipment. Engineering controls, alarms and good work practices are essential. Exposure can result in headache, nausea and irritation of the eyes, nose and respiratory tract. High concentrations can cause central nervous system depression, vomiting, dyspnea, cyanosis and pulmonary edema. Chemical burns may result from contact with inadequately aerated equipment or wrappings sterilized with ethylene oxide. This chemical is a suspect human carcinogen with respect to leukemia.

Every patient infection implies an employee exposure. Scabies can be a distressing condition for nursing and housekeeping staff. In addition to exposure to wound infection, having numerous microbiologic causes, nurses may be exposed to patients with such systemic diseases as tuberculosis, hepatitis B, varicella zoster, meningococcemia, cytomegalovirus and rubella.

Herpes simplex infections of the cuticle areas of the thumb and fingers are seen in nurses handling upper respiratory tract secretions, particularly in intensive care units, where patients are on ventilators.

Tuberculosis, while less common than in the past, continues to have a presence in active treatment hospitals. In Ontario, former T.B. sanatoria have taken on new functions, and new cases must be managed in general hospitals. Some of these patients are admitted as diagnostic problems, and, unless the disease is suspected, measures may not be taken to protect staff. More recently, tuberculosis has reappeared in association with human immuno–deficiency virus (HIV) infection and AIDS. The multidisciplinary team approach to patient care, especially in teaching hospitals, may result in multiple contacts in only a few days. With the involvement of investigational areas such as imaging and endoscopy, there are many opportunities for staff exposure to the unsuspected case. Therefore, while the overall prevalence of the disease is low, the threat to staff remains high. It is important to establish significant contact as opposed to casual exposure, since follow–up is prolonged and may lead to months of treatment with isoniazid.

The threat of exposure to hepatitis B and its attendant risks of developing acute and chronic hepatitis is a major concern for hospital staff who have contact with blood and other body fluids that have been shown to be infectious. Needlestick injuries are still common despite major efforts directed at proper disposal methods, including 'no recapping'. Hepatitis B immune globulin and

vaccines have been of great value, but they do not reduce the importance of employee awareness and good work practices. The acceptance of immunization against hepatitis B by all clinical and laboratory personnel is a goal still to be achieved.

The increasing prevalence of HIV infection and AIDS increases the concern that health-care workers may be exposed to infected blood and body fluids. Hospital personnel should consider all patients to be possibly infected with HIV and, to minimize the potential for transmission of infection, should conscientiously adhere to the principles and practice of universal precautions. Hospitals must have a specific HIV post-exposure protocol and ensure that all health-care workers at risk are aware of it.

Some types of protective equipment, such as the laboratory coat, have been in use in hospitals for so long that they are taken for granted and not recognized for their personal protective benefit. Surgical masks and gloves, introduced in the interest of patient protection, have assumed an equally important role in staff protection.

Soft Tissue Injuries

Lifting is associated with back problems among various hospital employee groups: nurses and orderlies, as well as stores, food services and housekeeping staff. These exertional strain incidents result in more than 40 per cent of a hospital's lost-time Workers' Compensation claims. Some patients are extremely obese, weighing more than 300 pounds, and, even with the assistance of mechanical devices for lifting, much patient care still requires manual handling. Sometimes unexpected patient action, as in falling or jerking, will elicit a spontaneous exertional effort by the nurse, orderly or physiotherapist. While this action will usually protect the patient, it may result in strain for the health-care worker. Correct posture for lifting, pushing, bending and reaching is always desirable, but the circumstances of patient care may preclude the application of proper body mechanics or the wait for help to arrive. In some persons with soft tissue conditions due to exertion or contusion, chronic pain

states emerge, reflecting psychosocial and lifestyle influences.

Lifting or straining causes increased intra-abdominal pressure, which may result in inguinal hernia through a weak area in the abdominal wall. Pain is usually the first symptom, and it may be accompanied by a noticeable bulge in the groin or upper thigh.

Puncture wounds occur in housekeeping, laundry and central supply staff as a result of careless disposal of needles, blades or other sharp items in garbage bags, in laundry and on used procedure trays. These punctures, usually on the hand but occasionally on the leg, are painful surprises to the recipient, who then expresses anger and resentment. These staff are at risk for infection and should be included in immunization programs.

Psychosocial Hazards

Hospital function is continuous, characterized by various combinations of shift work. Nurses on rotating shifts visit occupational health clinics more frequently than do their colleagues working regular shifts. This first group tends to seek medical attention more often for respiratory infections, influenza, upper and lower gastrointestinal symptoms and headaches. For night shift staff, sleep is interrupted by the normal noises of the day. Family and marital relationships may be strained.

Nurses face mentally and emotionally taxing patient-care situations, ranging from severe illness and sudden death in intensive care units to terminal illness and inevitable death in palliative care units. They must contend with patient, and occasionally visitor, hostility and display empathy while maintaining defensive strategies to preserve their own physical and mental well-being. Work overload due to staffing irregularities can have an impact on job satisfaction.

Nursing staff and medical staff may turn to medications for the symptomatic relief of their personal ills—physical and psychological. The strict control of narcotics has a protective effect, but many other medications are easily acquired and abused,

resulting in various degrees of chemical dependency and self-harm. The availability of specialty treatment areas and diagnostic facilities within the hospital contributes to the practice of self-referral to clinics, often for laboratory and imaging examinations. This practice results in sometimes needless, inadequate and inappropriate use of these services and promotes an unco-ordinated approach to the employee's own health.

Conclusion

Hospital philosophy has always acknowledged the undisputed first priority of patients, but this concept does not preclude the employees' right to a healthy and safe workplace. One thing would seem certain: the past practice of resources borrowed from medical staff is no longer acceptable as a response to occupational health issues in hospitals. These needs can be met only by expertise in occupational medicine, nursing, hygiene and rehabilitation.

Résumé

Les hôpitaux sont un ensemble complexe de services cliniques, de laboratoires et d'hôtellerie. Les divers risques de santé qui en résultent pour le personel exigent la mise en jeu des principes et méthodes modernes de médecine et de soins infirmiers, d'hygiène et de réadaptation professionnelle.

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A Multidisciplinary Approach for the Administration of Indoor Air Quality Questionnaires

Summary

This paper presents a protocol designed to ensure an appropriate strategy for administering an indoor air quality (IAQ) questionnaire to two subgroups. The roles of the occupational hygienist and nurse in administering the questionnaires at affected workplaces are described, as is the need for maintaining the results in confidence. The questionnaires are included as an appendix to the paper.

Key Words: indoor air quality, questionnaire, work environment, survey

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Introduction

An indoor air quality (IAQ) questionnaire is a useful tool for gathering information about the types of problems experienced in the indoor environment. It allows one to establish whether and to what extent IAQ problems exist. Different questionnaire designs have been used, but a lack of adequate direction in interpreting responses has proven a major drawback.

In 1988, an Ontario interministerial committee designed a questionnaire to be used as part of indoor air quality investigations.[1] In order to field test and validate its usefulness, the questionnaire was used in conjunction with routine investigations of the Health and Safety Support Services Branch of the Ministry of Labour. Preliminary results were reported at the fifth International Conference on Indoor Air Quality and Climate, which was held in Toronto, July 29 to August 3, 1990.[2]

Following this initial validation, it was recommended that the questionnaire be simplified in an effort to secure as meaningful results as possible and to improve the return rate.

Additional parameters were considered during further validation: the quality of feedback, the investigative value of the questions, and the appropriate timing of questionnaire administration with respect to the investigation. As well, some questions did not yield relevant data for evaluation and statistical analysis. A simplified new questionnaire was therefore developed.[3]

From the Ministry of Labour's perspective, it was decided that the questionnaires be administered by an occupational health nurse in order to ensure confidentiality of the information. The nurse also ensures that all questions are completed.

This paper outlines the protocol for administering the revised questionnaire, explains the roles of the hygienist and the nurse, and concludes with a section on data analysis. The questionnaire appears as an appendix at the end of the paper.

The questionnaire may be used as a screening tool prior to or in conjunction with the investigation, provided the questionnaire analysis is reviewed by the hygiene consultant before concluding the investigation. The consultant may decide on the timing of questionnaire administration as it relates to the investigation.

IAQ Questionnaire

The revised questionnaire is composed of two parts. Subgroup A includes questions pertaining to health and the working area, and Subgroup B includes questions pertaining to the work environment. This division was made to eliminate any introduced bias, e.g. symptomatic respondents may answer the environmental questions in a negative way. It is important, however, to note that the analysis of the returned data treats both parts as one entry. Accordingly, Subgroup A questionnaires should be distributed with Subgroup B questionnaires to different workers sharing the same location or adjacent space. A new coding system is provided on the top sheet to identify the matching pair for computer entry:

Code 1 identifies the location of the respondent;
Code 2 indicates the serial number (e.g. 1A);
Code 3 indicates the matching questionnaire (e.g. 1B).

Visit Protocol

1. The hygienist must draw a floor plan for the complainants' floor. The floor plan should identify the areas served by individual HVAC systems if more than one system exists. Various cubicles or offices, if they exist, should also be labelled according to the air supply source.
2. Before presenting the questionnaire, the nurse consultant must seek agreement from management and the joint health and safety committee for employees in the affected work

area. A control group (i.e. from another department/area, without complaints) should also be asked to complete the questionnaire. The size of the control group may be as low as 10 per cent of the complainants' group. A floor plan must also be drawn for the control floor.

3. Prior to distributing the questionnaires, the nurse and the hygienist, using the floor plan as a guide, must complete the coding on the top sheet to ensure that matching pairs of the questionnaires are given to workers at the same location.
4. Since the questionnaire is self-explanatory, no attempt should be made by either the nurse or the hygienist to explain or suggest answers to the questions.
5. Completed questionnaires are to be collected by the nurse immediately; completion should be ascertained, and the questionnaires must be treated as confidential information.
6. The completed questionnaires and the floor plan should be returned by the nurse to the biostatistician for data analysis.
7. If a comparison between air-sampling results and questionnaire findings is required, the hygienist must indicate on the floor plan the sites of sampling and the contaminants sampled. In reporting the levels, the location should be clearly identified as per the floor plan.

Data Analysis

The data required from the completed questionnaires may be analysed using chi-square statistics to provide the following:

1. The difference in frequency of health symptoms between the complainant group and the control group.
2. The association of reported health symptoms with perceived environmental factors, per stratified data analysis, with different subgroups sharing common characteristics (e.g. gender, similar work settings, age).
3. The association of health symptoms with different work settings (e.g. office vs. open area).

4. The association of air–sampling results with health symptoms and perceived environmental problems as per questionnaire data. Such analysis is possible only if the sampling locations correspond to the locations identified in Code 1 of the individual questionnaire.

It is to be noted that, in order to secure meaningful statistical results, a minimum of 20 completed questionnaires for each group must be obtained.

Résumé

La présente étude porte sur un protocole établi afin d'assurer la mise en place d' une stratégie appropriée pour superviser deux sous–groupes à qui on aura soumis un questionnaire relatif à la qualité de l'air dans les bâtiments. Les rôles des hygiénistes et des infirmières au travail concernant l'administration des questionnaires dans les lieux de travail touchés y sont décrits, tout comme l'importance de conserver les résultats en toute confidentialité. Les questionnaires sont placés en annexe de la présente étude.

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Indoor Air Quality Questionnaire – Subgroup A

This questionnaire is designed to help assess the quality of the indoor environment at the work site located: _____

_____

Your answers will remain confidential and they will be used for analysis only.

The questionnaire is collecting data on your **HEALTH AND WORKING AREA.** Questions are answered by checking the appropriate box ☑, or by filling in a blank_____. There is also space for additional comments.

We are grateful for your help in completing this questionnaire. Please check that you have answered **ALL** the questions before sealing the questionnaire in the confidential return envelope.

Hand the sealed envelope to _____, who will be in the office while you are completing the questionnaire.

Date: _____ Time: _____

FOR OFFICE USE ONLY

CODE 1 _____

CODE 2 _____

CODE 3 _____

DESCRIPTION OF JOB AND OFFICE

(1) My employment status:

Full time ..... ☐
Part time ..... ☐
Temporary ..... ☐
Other (specify) _____

(2) I have worked in this area since (year) ___, (month) ___, (day) ___.

(3) I work at the present location ___ (hours) a day.

(4) I am in:

a closed office ..... ☐
my own cubicle ..... ☐
an open area shared with others ..... ☐
Other (specify) _____

(5) What is the geographic location of your desk within the building?

East Side ☐ North East Corner ☐
West Side ☐ North West Corner ☐
South Side ☐ South East Corner ☐
North Side ☐ South West Corner ☐

(6) Are you sitting within 3.7 metres (12 feet) of a window?

YES ☐ NO ☐

(7) Can the window be opened?

YES ☐ NO ☐

(8) Within 10 metres (approximately 33 feet) of your work location is there:

a typewriter? ..... YES ☐ NO ☐
a photocopying machine? ..... YES ☐ NO ☐
a keyboard with a video display screen ..... YES ☐ NO ☐
(e.g., VDT, CRT, data or word processor)?
a printer? ..... YES ☐ NO ☐
a teletype or fax machine? ..... YES ☐ NO ☐
a posting machine? ..... YES ☐ NO ☐
Other (specify) _____

DURING THE PREVIOUS WEEK WHILE WORKING IN YOUR AREA	
Check "YES" if symptoms interfered with work	Check the symptoms which have given you trouble
(9) Nasal symptoms YES ☐ ⇨ NO ☐ ⇩	Nosebleeds ☐ Congestion ☐ Sinus problems ☐ Sneezing ☐ Runny nose ☐ Dry nose ☐ Other (specify) _____
(10) Throat symptoms YES ☐ ⇨ NO ☐ ⇩	Sore throat ☐ Dry cough ☐ Other (specify) _____
(11) Eye symptoms YES ☐ ⇨ NO ☐ ⇩	Redness ☐ Watering ☐ Burning ☐ Puffiness ☐ Dryness ☐ Irritation ☐ Blurred vision ☐ Other (specify) _____
(12) Contact Lens Wearer YES ☐ NO ☐	
Problems related to wearing contact lens YES ☐ ⇨ NO ☐ ⇩	problems with: Cleaning ☐ Deposits ☐ Discomfort ☐ Pain ☐ Other (specify) _____

(13) Skin Problems YES ☐ ⇨ NO ☐ ⇩	Dryness ☐ Flaking ☐ Rash ☐ Irritation ☐ Other (specify) _____
(14) Aches and Pains YES ☐ ⇨ NO ☐ ⇩	Headache ☐ Backache ☐ Muscle/Joint pain ☐ Other (specify) _____
(15) General Complaints YES ☐ ⇨ NO ☐ ⇩	Drowsiness ☐ Dizziness ☐ Faintness ☐ Difficulty in concentration ☐ Other (specify) _____
(16) Other symptoms YES ☐ ⇨ NO ☐ ⇩	Breathing ☐ Digestive ☐ Menstrual ☐ Other (specify) _____

(17) Were you ever absent because of any health problem(s) that you feel may have been caused or aggravated by working at your present location?

YES ☐ NO ☐

If YES, state the health problem: _____

_____

_____

_____

(18) Did you seek medical treatment because of any health problem(s) caused by working at your present location?

YES ☐ NO ☐

(19) Do you have allergies?

YES ☐ NO ☐

If YES, allergic to: _____

_____

_____

(20) Are you taking any prescribed medication for any symptoms you mentioned?

YES ☐ NO ☐

GENERAL INFORMATION

(21) Age (in years) _____

(22) Sex: Male ☐

Female ☐

ARE THERE ANY FURTHER COMMENTS WHICH YOU WOULD LIKE TO MAKE?

_____

_____

_____

_____

THANK YOU FOR FILLING IN THIS QUESTIONNAIRE

Indoor Air Quality Questionnaire – Subgroup B

This questionnaire is designed to help assess the quality of the indoor environment at the work site located: _____

_____

Your answers will remain confidential and they will be used for analysis only.

The questionnaire is collecting data on your **WORKING ENVIRONMENT.** Questions are answered by checking the appropriate box ☑, or by filling in a blank _____. There is also space for additional comments.

We are grateful for your help in completing this questionnaire. Please check that you have answered **ALL** the questions before sealing the questionnaire in the confidential return envelope.

Hand the sealed envelope to _____, who will be in the office while you are completing the questionnaire.

Date: _____ Time: _____

FOR OFFICE USE ONLY

CODE 1 _____

CODE 2 _____

CODE 3 _____

DESCRIPTION OF JOB AND OFFICE

(1) My employment status:

Full time ..... ☐
Part time ..... ☐
Temporary ..... ☐
Other (specify) _____

(2) I have worked in this area since (year) ___, (month) ___, (day) ___.

(3) I work at the present location ___ (hours) a day.

(4) I am in:

a closed office ..... ☐
my own cubicle ..... ☐
an open area shared with others ..... ☐
Other (specify) _____

(5) What is the geographic location of your desk within the building?

East Side ☐ North East Corner ☐
West Side ☐ North West Corner ☐
South Side ☐ South East Corner ☐
North Side ☐ South West Corner ☐

(6) Are you sitting within 3.7 metres (12 feet) of a window?

YES ☐ NO ☐

(7) Can the window be opened?

YES ☐ NO ☐

(8) Within 10 metres (approximately 33 feet) of your work location is there:

a typewriter? ..... YES ☐ NO ☐
a photocopying machine? ..... YES ☐ NO ☐
a keyboard with a video display screen ..... YES ☐ NO ☐
(e.g., VDT, CRT, data or word processor)?
a printer? ..... YES ☐ NO ☐
a teletype or fax machine? ..... YES ☐ NO ☐
a posting machine? ..... YES ☐ NO ☐
Other (specify) _____

WORKING ENVIRONMENT: Please record your general assessment of the working environment at your present location.

Check "YES" if disturbing	Check the aspects which are disturbing
(9) Noise YES ☐ ⇨ NO ☐ ⇩	Nearby conversation ☐ Lighting ☐ Ventilation system ☐ Office equipment ☐ Other (specify) _____
(10) Ventilation YES ☐ ⇨ NO ☐ ⇩	Temperature ☐ Humidity ☐ Air movement ☐ Other (specify) _____
(11) Lighting YES ☐ ⇨ NO ☐ ⇩	Too bright ☐ Not bright enough ☐ Glare, flicker ☐ Other (specify) _____
(12) Others YES ☐ ⇨ NO ☐ ⇩	Specify _____ _____ _____ _____ _____

(13) If there is a smell in your area, how would you describe the smell:

(a) The smell resembles:
- Glue ..... ☐
- Vinegar ..... ☐
- Alcohol ..... ☐
- Ammonia ..... ☐
- Propane ..... ☐
- Gasoline ..... ☐
- Perfume ..... ☐
- Other (specify) _____

(b) It smells:

Smoky ..... ☐

Dusty ..... ☐

Musty ..... ☐

Stale ..... ☐

Other (Specify_____

(c) In your opinion, where is the smell coming from?

_____

_____

_____

(14) Do you use any of the following in your work location?

(a) a desk lamp ..... ☐

(b) a fan ..... ☐

(c) a heater ..... ☐

(d) a humdifier ..... ☐

(e) an ion generator ..... ☐

(f) an air cleaner ..... ☐

(g) personal care products (e.g. hand cream, hairspray) (please specify what products) ☐

(h) no items ..... ☐

(15) Have you any control over your work location?

(a) Ventilation ..... ☐

(b) Temperature ..... ☐

(c) Humidity ..... ☐

(d) Lighting ..... ☐

(16) Is there a smoking room near your working location?

YES ☐ NO ☐

If YES

(17) How far are you away from smoking room _____(feet)

GENERAL INFORMATION

(18) Age (in years)_____

(19) Sex: Male ☐

Female ☐

ARE THERE ANY FURTHER COMMENTS WHICH YOU WOULD LIKE TO MAKE?

_____

_____

_____

_____

THANK YOU FOR FILLING IN THIS QUESTIONNAIRE

Book Reviews

Toxicological Chemistry: A Guide to Toxic Substances in Chemistry

Stanley E. Manahan

Lewis Pulbishers, Chelsea, Michigan, 1989

Stanley Manahan is a professor of chemistry at the University of Missouri–Columbia, and his primary research and professional activities have been in the area of environmental chemistry. He has undertaken the daunting task of bridging the gap between toxicology and chemistry, with the emphasis on the latter. Other texts tend to delve deeply into all aspects of toxicology, often at a level that does not suit the practising occupational hygiene professional. Such practitioners, who represent a variety of backgrounds, will find this text useful in furthering their knowledge.

The text is divided into 14 chapters, with the first two describing the fundamental terms and concepts in toxicology and chemistry. In Chapter 3, Professor Manahan combines the two topics to discuss the routes of exposure to toxic substances and the effects of such exposure, using several examples of chemical toxins. Any chapter on metabolism and biotransformation presents a challenge, since the depth of coverage of the topic is difficult to gauge. The author has chosen to be brief but all–encompassing, offering in Chapter 4 more than ample information for the journeyman hygienist.

Chapter 5 includes more advanced chemistry, to help in the comprehension of the balance of the text. Chapters 6 through 13 are treatises of eight classes of toxic chemicals: organometallics and organometalloids; toxic inorganic compounds; toxic organic compounds and hydrocarbons; oxygen–containing organic compounds; nitrogen–containing organic compounds; organohalides; sulfur–containing organic compounds; and phosphorous–containing organic compounds.

All chapters contain useful, albeit cursory, information on most if not all classes of toxins; the exhaustive chapter on organometals is particularly well done. The author's dilemma was to cover a broad topic in sufficient detail to make his work useful, and despite the relative brevity of discussion on many chemicals, the book is still more than 300 pages. More attention given any topic would have made the text uncomfortably long.

The final chapter, which deals with toxic natural products, makes for a welcome break, although, at the risk of making the book overly long, the author could have devoted more pages to the relatively high daily exposure to 'natural' chemical toxins. If explored properly, a discussion of their consequences would provide the health professional with material to present in public forums when explaining the relative risk of exposure to 'manmade' chemical toxins.

Since the text deals with chemicals, there is no real information on silica, asbestos and other toxic dusts. Thus, the book should not be seen as a complete toxicology reference. Written in an easy–to–read style, the text would do well as an introduction to toxicological chemistry for any occupational health professional.

M.A. Nazar

Raynaud's Phenomenon

Jay D. Coffman

Oxford University Press, 1989

The incidence of Raynaud's phenomenon in the community is thought to be at least 10 per cent, and some recent reports have suggested that it may be as high as 20 per cent. This monograph then is a timely review to assist physicians to better understand the cause as well as the state–of–the–art treatment available.

The author helpfully reminds us of the anatomy and physiology of finger circulation; the clinical picture of primary Raynaud's disease and its prognosis; the pathophysiology; and the secondary causes. The author quite rightly states 'patients with Raynaud's phenomenon should be investigated for

an underlying disease only when the history, physical examination, or routine laboratory tests indicate that they may be present. A complete work–up for all secondary causes would be expensive and not cost effective.' The numerous secondary causes, including repetitive trauma from the use of vibratory tools, are adequately reviewed, and the chapter on treatment is timely. Again the advice is sound: 'Most patients with Raynaud's phenomenon, primary or secondary, should undergo a trial of conservative treatment before drug therapy is considered. If conservative therapy fails to give the patient adequate relief, drug treatment can be recommended.'

Each chapter concludes with a summary that the busy practitioner will welcome, and the book will be useful to medical students as well as to family practitioners. This book deserves to be widely read, and I am happy to have it in my library.

P.L. Pelmear

Recent Publications Available from the Ministry of Labour

Revisions

Occupational Health and Safety Act, as amended by Bill 208

A Guide to the Occupational Health and Safety Act

A Guide for Joint Health and Safety Committees

Unvented Gas-Fired Infra-Red Heaters: Engineering Data Sheet

List of Occupational Health and Safety Consultants in Ontario, 1990/91 Edition

Additions

Robots and Robot Systems: Engineering Data Sheet

Industrial Information Alerts:

- Bumper Jack Failure: Fatality
- Unguarded Cut-off Saw: Critical Injury
- Vehicle Fires: Potential Hazards
- Collapse of Bales: Critical Injury
- Electrocution: Fatality
- Arm Amputations: Critical Injuries
- Brakes: Fatalities and Critical Injuries

For further information, contact:

Information and Administrative Services
Edit and Control Section
(416) 326-7744

News and Notes

The 18th Semi-annual Intensive Workshops in Industrial Hygiene, University of Toronto

September 23–27, 1991

These workshops are designed for those who wish to increase their knowledge in their specialization in occupational hygiene; for those who feel the need for a general refresher course; and for recent entrants to the hygiene field who wish to broaden their background knowledge of occupational hygiene.

The panel of lecturers includes Dr. John Steel, Dip. Occ. Hyg., Department of Occupational Health and Hygiene, Medical School, University of Newcastle–upon–Tyne, United Kingdom; personnel from the Ontario Ministry of Labour; and faculty and staff of the University of Toronto. The expertise of the speakers covers all relevant aspects of hygiene, risk assessment, human factors engineering, toxicology, occupational medicine, and occupational health and safety legislation. A hands–on equipment demonstration and monitoring round–table are included in the workshop.

The Industrial Hygiene and Hazard Control Engineering Program of the University of Toronto presents the workshops bi–annually. Professor J.W. Smith, Ph.D., C.Eng., C.I.H., chairman of the Department of Chemical Engineering and Applied Chemistry, is the program coordinator.

Enquiries should be addressed to:

Julie Mendonça
Department of Chemical Engineering
Applied Chemistry
200 College Street
University of Toronto
Toronto, Ontario
M5S 1A4
(416)978–6615

Occupational Health Chemists

Managers of occupational health laboratories in the province of Ontario have started a process to form an association. This association's principal aims are to run proficiency–testing programs, to hold regular technical meetings and to maintain professional practices as chemists. A steering committee and a proficiency–testing subcommittee have met several times to provide direction for the accomplishment of these goals. For the present, the association most likely will be constituted as a section of the Association of the Chemical Profession of Ontario, which was formed by the legislature in 1984.

If you have not been contacted about this process and are interested in this association and its proficiency–testing programs, please direct enquiries to:

Mark A. Nazar, Ph.D., C.I.H., R.O.H.
Chief Scientist
Ministry of Labour
Occupational Health Lab
101 Resources Road
Weston, Ontario
M9P 3T1
Telephone: (416)235–5958
Fax: (416)235–6024

Coming Events

September 4–8, 1991	Health Buildings: IAQ '91. WASHINGTON, D.C., U.S.A. For further information, contact: Jim Norman, Manager of Technical Services, ASHRAE, 1791 Tullie Circle NE, Atlanta, Georgia, U.S.A., 30329.
September 10–12, 1991	8th International Symposium, Epidemiology in Occupational Health, PARIS, FRANCE. For further information, contact: Convergence 120, Avenue Gambetta, 75020, Paris, France.
September 16–18, 1991	Safety & Health throughout the Planning and Construction Process. 13th International Colloquium. International Section of the ISSA for the Prevention of Occupational Risks in the Construction Industry. BRUSSELS, BELGIUM. For further information, contact: Conference Secretariat, NAVB–CNAC, Boulevard Poincaré, 70, B–1070, Brussels, Belgium.
September 16–20, 1991	Ventilation '91: 3rd International Symposium on Ventilation for Contaminant Control, CINCINNATI, OHIO, U.S.A. For further information, contact: ACGIH, 6500 Glenway Ave., Building D7, Cincinnati, Ohio, U.S.A., 45211–4438. Telephone: William Kelley (513) 661–7881.

September 16–20, 1991	7th International Symposium on Inhaled Particles. EDINBURGH, SCOTLAND. For further information, contact: Dr. A. Robertson, Organizing Secretary, British Occupational Hygiene Society, 7th International Symposium on Inhaled Particles, Institute of Occupational Medicine, Roxburgh Place, Edinburgh EH8 9SU Scotland.
September 21–23, 1991	3rd International Conference on Education & Training in Occupational Health & 11th UEOEH International Symposium. KITA–KYUSHU, JAPAN. For further information, contact: University of Occupational Environmental Health, Toshiteru Okubo, MD, Yahatanishi–ku, Kita–Kyushu 807, Japan.
September 22–24, 1991	Air & Waste Management Association (Ontario Section), Annual Fall Meeting: Environmental Management Initiatives: Focus on Municipal and Industrial Experiences. ORANGEVILLE, ONTARIO, CANADA. For further information, contact: Stephen Toplak, Telephone: (416) 735–0035.
October 15–17, 1991	Profit from Prevention. International Occupational Health & Safety Convention 1991. MELBOURNE, AUSTRALIA. For further information, contact: Sue Wood & Associates, 1st Floor, 387 Malvern Rd., South Yarra, Victoria, Australia, 3141. Telephone: (03)824 0022. FAX: (03)826 0771.
October 26, 1991	Scientific Basis of Back Injury Symposium, Queen's University, KINGSTON, ONTARIO, CANADA. For further information, contact: Dr. Joan Stevenson, Ergonomics Research Group, School of P.H.E., Queen's University, Kingston, Ontario, Canada, K7L 3N6. Telephone: (613) 545–6288. FAX: (613) 545–2009.

October 28–November 1, 1991	ACOM State–of–the–Art Conference. ST. LOUIS, MISSOURI, U.S.A. For further information, contact: ACOM, 55 W. Seegers Rd., Arlington Heights, Illinois, U.S.A., 60005.
October 29–November 1, 1991	International Symposium on Air Sampling Instrument Performance: Applications, Technology, Criteria & Standards. RALEIGH, NORTH CAROLINA, U.S.A. For further information, contact: Air Sampling Instrument Performance Symposium, c/o ACGIH, 6500 Glenway Ave., Building D7, Cincinnati, Ohio, U.S.A., 45211–4438.
October 29–November 1, 1991	30th Hanford Symposium on Health & the Environment. Current Topics in Occupational Health. RICHLAND, WASHINGTON, U.S.A. For further information, contact: Ray W. Baalman, Manager, Planning & Communications, K1–50, Life Sciences Center, Battelle, Pacific Northwest Laboratories, Richland, Washington, U.S.A., 99352. Telephone: (509) 375–3665. FAX: (509) 375–3686.
November 1991	1991 Annual Meeting of the International Society of Exposure Analysis. ATLANTA, GEORGIA, U.S.A. For further information, contact: Gerry Akland (MD–75), Environmental Protection Agency, Research Triangle Park, North Carolina, U.S.A., 27711. FAX: (919) 541–7588.
November 1991	1st International Symposium on Global Warming and Human Health. KHARTOUM, SUDAN. For further information, contact: Dr. Moneim Attia, IHSRC, P.O. Box 2020, Khartoum, Sudan.

November 10–14, 1991	American Public Health Association: 119th Annual Meeting. ATLANTA, GEORGIA, U.S.A. For further information, contact: Bob Johnson, APHA, 1015 15th St. N.W, Washington, D.C., 20005, U.S.A. Telephone: (202) 789–5672. FAX: (202) 789–5661
November 17–20, 1991	Asian Association for Occupational Health: Asian Conference on Occupational Health. BANGKOK, THAILAND. For further information, contact: Professor Malinee Wongphanich, Occupational Health Dept., Building 3, Faculty of Public Health, Mahidol University, Bangkok, Thailand. Telephone: 662 246–1258, extension 259/260/261. FAX: 662 246–7765.
December 3–7, 1991	International Conference on Human Environment System. TOKYO, JAPAN. For further information, contact: Yoshikatsu Kawashima, Secretary General, ICHES, c/o Dept. Mechanical Engineering, Yokohama, National University, Tokiwadai 156, Hodogaya–ku, Yokohama 240, Japan. Telephone: 81 45 335 1451, extension 2666.
May 17–22, 1992	8th World Congress of the International Radiation Protection Association. MONTRÉAL, QUEBÉC, CANADA. For further information, contact: Dr. J.P. Gauvin, 2155 Rue Guy, Bureau 820, Montréal (Québec) Canada, H3H 2R9.
May 24–27, 1992	Futuresafe 92: Operating Safe Environment. National Safety Council of Australia. ADELAIDE, SOUTH AUSTRALIA. For further information, contact: NSCA Congress & Exposition, c/o International Convention Management Services, P.O. Box 8102, Hindley St., Adelaide, South Australia, 5000. Telephone: (08)210–6776. FAX: (08)212–5101.

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September 18–20, 1992	Harvard School of Public Health: Indoor Air Quality. BOSTON, MASSACHUSETTS, U.S.A. For further information, contact: Office of Continuing Education, Dept. B, Harvard University School of Public Health, 677 Huntingdon Ave., Boston, Massachusetts, 02115, U.S.A.

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